NEUROSCIENCE

Correction for "A conserved salt bridge critical for $GABA_A$ receptor function and loop C dynamics," by Srinivasan P. Venkatachalan and Cynthia Czajkowski, which appeared in issue 36, September 9, 2008, of *Proc Natl Acad Sci USA* (105:13604–13609; first published August 29, 2008; 10.1073/pnas.0801854105).

The authors note that in Fig. 1 the alignment of the nicotinic receptor α1 subunit sequences (*Torpedo californica* and rat) in the loop C region with the $GABA_A$ receptor β subunit sequences aligns Y190 and T191 of the former with K196 and K197 of the latter. The alignment should align H186 and W197 of the nicotinic receptor with K196 and K197 of the $GABA_A$ receptor. The nicotinic sequence that reads VYYTCCPD 195 should read WKHW..YT 191. The error does not alter the conclusions of this article. The corrected figure and its legend appear below.

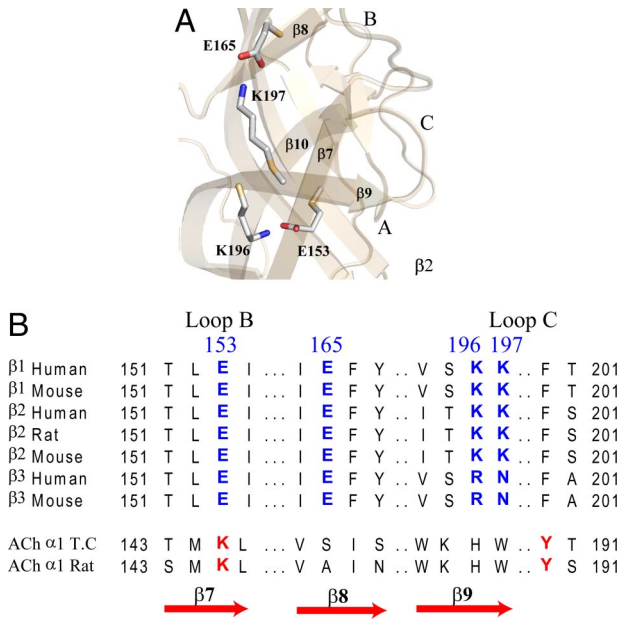

Fig. 1. Model of $GABA_AR$ extracellular N-terminal domain based on AChBP (ligand bound). (*A*) Charged residues in the β2 subunit (E153, E165, K196, and K197) that might be involved in regulating movement of loop C via electrostatic interactions are shown. Binding site loops A, B, and C are marked. (*B*) Sequences of various $GABA_AR$ β-subunits highlighting conserved charged residues (blue). Aligned residues in the nAChR α-subunit from *Torpedo californica* and *Rattus norvegicus* are also shown. Residues suggested to form a salt bridge important for stabilizing the open state of nAChR (Mukhtasimova *et al.*) (11) are colored red.

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MEDICAL SCIENCES

Correction for "Abnormal integrin-mediated regulation of chronic myelogenous leukemia $CD34^+$ cell proliferation: BCR/ABL up-regulates the cyclin-dependent kinase inhibitor, $p27^{Kip}$, which is relocated to the cell cytoplasm and incapable of regulating cdk2 activity," by Yuehua Jiang, Robert C. H. Zhao, and Catherine M. Verfaillie, which appeared in issue 19, September 12, 2000, of *Proc Natl Acad Sci USA* (97:10538–10543; first published September 5, 2000; 10.1073/pnas.190104497).

The authors wish to note the following: "We inadvertently inserted the Western blot from a different experiment (Western blot for Cdk2, following immunoprecipitation of Cdk2 from a different experiment), instead of the correct Western blot for Cdk4, following immunoprecipitation of Cdk4 for the study described in Fig. 3. The correct Western blot for Cdk4-IP/WB: Cdk4 has now been inserted as lane 3 of the figure. In addition, we have added a black line in the Cdk2 kinase assay as well as in the Cdk4-IP/WB: Cdk4 Western blot to indicate that a blank lane was removed. We apologize for any inconvenience this may have caused." This error does not affect the conclusions of the article. The corrected figure and its legend appear below.

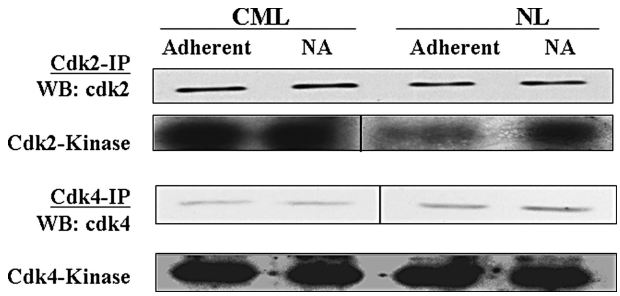

Fig. 3. Elevated levels of $p27^{Kip}$ do not inhibit cdk2 or cdk4 activity. Proteins were extracted from 5–10 $\times$ 10^6 FN-A, FN-NA, PLL-A (not shown), and PLL-NA (not shown) CML or NL $CD34^+$ cells. cdk2 and cdk4 were immunoprecipitated from 500 μg protein by using anti-cdk2 or anti-cdk4 antibodies, or control IgG and protein-G-agarose beads, separated by SDS/PAGE and blots probed with anti-cdk2 or anti-cdk4 and goat anti-mouse HRP antibodies. cdk2 and cdk4 activity was assayed by adding 5 μg histone or GST-Rb and 10 μCi [r-^{32}P] to immune complexes. Reaction products were resolved by SDS/PAGE, and the gel was exposed to X-ray film. A representative example of three separate experiments is shown.

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CORRECTIONS